Royal Naval dockyards asbestosis research project: nine-year follow-up study of men exposed to asbestos in Devonport Dockyard[1]

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Summary: Progression of asbestos-related disease was assessed in a group of 253 dockyard workers examined in 1966 and 1975. Despite the almost complete protection from exposure to asbestos since 1966, radiographic parenchymal abnormalities increased and occurred more frequently during the next 9 years in those men who had been more heavily exposed to asbestos. Lung function values were lower in those most heavily exposed and were declining at a faster rate than in those with less dust exposure. The most sensitive lung function index was the transfer factor.

Those men with persistent crackles in 1966 exhibited a restrictive pattern of lung function, whereas an obstructive pattern was seen in men with wheezes in 1966. The men with irregular small opacities of category 1/1 or more or with diffuse pleural change in 1966 and who survived to 1975 had worse lung functions than any other groups.

Progression of disease was greater for smokers than non-smokers, with those who gave up smoking between 1966 and 1975 suffering the greatest changes. This latter group showed most increase in small opacities and included almost all new cases of diffuse pleural changes. They also showed the greatest declines in forced expiratory volume and forced vital capacity.

Introduction

In 1965 it was discovered that 10% of laggers working at Devonport Dockyard were suffering from asbestosis. Further investigation showed that 8 of 17 asbestos sprayers were also suffering from the disease. These findings led to a detailed study of 420 men at Devonport Dockyard (Harries 1970), which confirmed the work of others (Selikoff *et al.* 1965, Elmes 1966, Sheers & Templeton 1968) that men working as insulators in shipyards were exposed to asbestos dust and were being affected by asbestos-related diseases. It also showed that other men working with or near asbestos workers were contracting asbestos disease.

The study examined the effects of exposure on clinical, radiological and lung function findings in a population consisting of all living sprayers who could be traced, all working laggers, a group of men working as asbestos storemen, sailmaker laggers using asbestos cloth, and masons using asbestos cement and flooring material as well as a random sample of men working afloat in a variety of trades thought to be intermittently exposed to asbestos.

The results showed that the clinical, radiographic and lung function findings were interrelated and associated with the intensity and duration of exposure to asbestos. They demonstrated that men with radiographic pleural abnormalities in addition to their parenchymal abnormalities had worse lung function than those without these changes. This has now been confirmed by Becklake *et al.* (1970) and Lumley (1977).

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The survivors of this population chosen in 1966 have been used to study the progression of asbestos disease in a group which was effectively removed from further exposure to asbestos from the start of the 1966 survey.

Methods

Population

The population studied comprised the survivors of the group of 420 men selected for study in 1966 and reported by Harries (1970). In that study 402 of the 420 were examined, but 33 of those examined were excluded from the analyses because of other chest diseases or exposure to other dusts, which left 369 men for whom data were available for analysis. Full details of the reasons for exclusion were given by Harries (1970). In the nine years between the first study and the present survey 43 men died, leaving 326 men alive. Of this population, 268 (82%) men were re-examined in 1975 and 58 were not examined. Sickness prevented 11 men from being examined, 34 had left the Plymouth area or their current address was unknown and 13 men refused to participate. No lung function measurements were done on 8 men in 1975 and incomplete lung function on a further 6. The 1975 radiograph was lost for one man, leaving 253 men with complete data. Table 1 shows the numbers of men selected for examination, those examined in the first study, and also those re-examined in 1975.

Chest radiography

The chest radiographs were taken by the same Medical Research Council Pneumoconiosis Unit Mobile Radiographic Unit that took the films for the first survey. A standardized technique was used to take posteroanterior radiographs on 400 mm × 400 mm films and development was by automatic processor.

The films of the men examined in 1966 and 1975 were placed in random order and read independently by five readers (J C Gilson, P G Harries, K P S Lumley, G Sheers, F A F Mackenzie) to a slightly extended version of the ILO U/C classification of radiographs for pneumoconiosis (International Labour Office 1972). This extension permitted the separate recording of diffuse pleural thickening from pleural plaques based on the outline and appearance of the pleural shadows (Rossiter & Harries 1979).

Clinical examination

Each subject was interviewed by the same observer (PGH) in 1966 and 1975 and the Medical Research Council standard questionnaire on respiratory symptoms was completed together with a full occupational history. Physical signs were recorded as being absent or present, and included assessment of general physique, crackles, wheezes and finger clubbing. Height and weight were measured without shoes or top coats.

Lung function measurements

Forced vital capacity (FVC) and one-second forced expiratory volume (FEV_1) were measured using the same McDermott dry spirometer as was used in the 1966 study, and by the same observer. The spirometer timing was checked several times during the study as well as at the beginning and the end of the survey. Volumetric calibration was also carried out. All volumes were corrected to BTPS.

Measurements of single-breath carbon monoxide transfer factor (TL) were made in duplicate using a Morgan Mark IV Resparameter with a 5-minute interval between measurements. The same resparameter was used for the measurement of lung volumes by the helium rebreathing method. The spirometer bell was calibrated against volumetric glassware and the kymograph speed calibrated using an accurate stopwatch. Measurements of volumes and breath-hold times from the traces were corrected accordingly. Gas sample concentrations were corrected for sample bag dead space and for the removal of CO_2 and water vapour. The helium and carbon monoxide analysers deviated from linearity by less than 0.5% of maximum measured concentration so that no linearity corrections were applied. Appropriate corrections were made for room temperature and pressure.

Table 1. Population selected for study in 1966 and numbers included in analysis in 1966 and 1975

Occupation groups	Selected for 1966 study	Examined in 1966	Included in 1966 analysis●	No. from 1966 analysis alive in 1975	Survivors with incomplete data	Survivors with complete data
Group I: Asbestos sprayers	55	52	50	37	4	29
Group II: Laggers	108	107	98	86	4	72
Group III: Asbestos storemen, sailmaker laggers, 'asbestos' masons	58	57	45	37	3	28
Group IV: Various trades intermittently exposed in neighbourhood of groups I, II and III	198	185	176	166	4	124
Total	420	402	369	326	15	253

●Reasons for exclusion, together with data on these men are given in the original report (Harries 1970)

Table 2. Prevalence (%) of symptoms and signs in 1975 by occupation group

Symptoms or signs	Occupation group I (29 men)	II (72 men)	III (28 men)	IV (124 men)	All subjects (253 men)
Breathlessness ●	62%	26%	11%	8%	20%
Crackles	48%	19%	7%	7%	15%
Wheezes	7%	10%	4%	12%	10%
Finger clubbing	21%	8%	0%	2%	6%

● MRC Grade 3: breathless walking with people of same age on level ground

Table 3. Numbers of men with symptoms or signs in the two surveys (1966 and 1975)

	Absent in 1966		Present in 1966	
	Absent 1975	Present 1975	Absent 1975	Present 1975
Breathlessness grade 3 or over	198	33	5	17
Crackles	198	12	16	27
Wheezes	214	13	14	12
Definite clubbing	239	11	0	3

Results

Symptoms and signs

The prevalence rates for breathlessness (MRC Grade 3 or worse), crackles, wheezes and finger clubbing are given in Table 2. There were distinct trends for the prevalence rates for breathlessness, crackles and finger clubbing to be highest in occupational groups I and II, who had been more heavily exposed to asbestos, and lowest in groups III and IV. This trend was not seen for wheezes, which were significantly related to smoking ($P<0.05$), having been detected in 13% of the smokers and none of the non-smokers. These patterns were similar to those seen in the original study.

Changes in the numbers of men with recorded symptoms and signs are given in Table 3. Allowing for subjective variation the overall pattern for breathlessness showed a considerable increase in the numbers of men with this complaint. The effects of observer variation as well as the intermittent occurrence of crackles and wheezes probably explained the interchange in the presence or absence of these signs between the two surveys. Finger clubbing, when recorded as 'marked' or 'definite' was the only physical sign to show an increased prevalence, though some of the cases regarded as positive in 1975 were recorded as 'doubtful' or 'early' clubbing in 1966. It should be remembered that several of the men with these signs in 1966 died in the meantime and were not included in these data.

Smoking habits

There were no big differences in the proportion of smokers between occupation groups in the original study, and Table 4 shows the numbers of smokers in 1966 and the 1975 smoking habits. One non-smoker in 1966 had become a smoker by 1975, and 2 ex-smokers in 1966 had resumed smoking by 1975, while 27 smokers in 1966 had given up smoking by 1975. Those who gave up smoking were distributed evenly by occupation group and there were still no big differences between the groups in the proportions of smokers and non-smokers.

Table 4. Smoking habits in 1966 and 1975

		1975 smoking habit		
1966 smoking habit		Non-smokers	Ex-smokers	Smokers
Non-smokers	34	33	0	1
Ex-smokers	50	0	48	2
Smokers	169	0	27	142
Total	253	33	75	145

Radiographic abnormalities

The classification of radiographs by occupation group is shown in Table 5 for the 1966 and the 1975 films. There has been a marked increase in prevalence of small opacities of category 1/1 or more since the 1966 survey, particularly among the sprayers and laggers (occupation groups I and II). Table 6 shows that this increase in prevalence was greatest among the 27 men who gave up smoking between 1966 and 1975. However, the prevalence was also increased among those who continued to smoke, but not among the remaining men.

Diffuse pleural thickening was also much more prevalent in 1975 than in 1966, particularly among the sprayers and laggers (Table 5) and Table 6 shows that this increase was almost exclusively limited to those who gave up smoking after 1966. There was not much increase in the prevalence of pleural plaques, although 7 of the 35 with plaques in 1966 developed diffuse pleural changes by 1975. Smokers showed the greatest increase in the prevalence of pleural plaques, from 10% in 1966 to 23% in 1975. Pleural calcification was slightly more common in 1975 but most of the overall increase in pleural abnormalities was due to the increase in diffuse thickening, particularly among those who gave up smoking between the two surveys.

Table 5. Radiographic abnormalities in 1966 and 1975 by occupation group (I–IV)

Abnormality	Year	I (29 men)	II (72 men)	III (28 men)	IV (124 men)	All (253 men)
Small opacities (category 1/1 +)	1966	7%	3%	4%	1%	2%
	1975	33%	19%	0%	4%	11%
Diffuse pleural thickening	1966	3%	1%	0%	3%	2%
	1975	17%	10%	0%	7%	13%
Pleural plaques	1966	24%	14%	4%	14%	14%
	1975	20%	26%	7%	23%	17%
Pleural calcification	1966	3%	1%	0%	1%	1%
	1975	7%	4%	0%	5%	4%
All pleural changes	1966	28%	15%	4%	17%	16%
	1975	38%	36%	7%	31%	30%

Table 6. Prevalence of small opacities and diffuse pleural thickening in 1966 and 1975 by smoking habit

	Number of men	Small opacities (category 1/1 or more)			Diffuse pleural thickening		
		Present 1966	Present 1966, absent 1975	Present 1975	Present 1966	Present 1966, absent 1975	Present 1975
Smokers in 1975	145	2%	1%	12%	2%	0%	5%
Smokers in 1966 but ex-smokers in 1975	27	4%	0%	33%	7%	4%	30%
Other ex-smokers in 1975	48	2%	2%	4%	2%	2%	8%
Non-smokers in 1975	33	3%	3%	3%	0%	0%	3%
	253	2%	1%	11%	2%	1%	13%

Lung function

All lung function results for 1966 have been standardized to the overall average age (42) and height (1.706 metres) in 1966 using the equations of Cotes (1965) for normal men. Similarly, the 1975 results have been standardized to average age 51 and height 1.709 metres.

Mean standardized values for the lung function tests for the four occupation groups are shown in Table 7. There were consistent differences between occupation groups, with lowest values for forced expiratory volume, forced vital capacity, total lung capacity and transfer factor in occupation groups I and II and highest in occupation group IV. Further, the decline in lung function also tended to be greatest in occupation group I, although the average annual changes were not very different from the expected rates.

To clarify how much these differences could be attributed to exposure, as assessed by occupation group, and how much to other factors, standard analyses of each lung function index were carried out to estimate the contributions of occupation group, smoking habit, physical signs in 1966 and radiographic abnormalities in 1966 to the variation in lung function. Figures 1 to 4 show the results of these analyses.

Figures 1 and 2 show that total lung capacity was lowest in occupation group II and highest in IV in both 1966 and 1975 even after allowing for the relations to smoking, physical signs and radiographic abnormalities. However, the greatest average reduction in total lung capacity occurred among the 36 men with persistent crackles in 1966. The 195 men without small opacities had the highest values.

Table 7. Lung function by occupation group

	Year	Occupation group I (29 men)	II (72 men)	III (28 men)	IV (124 men)	Total (253 men)	Average change per annum
Forced expiratory vol (1 sec) (litres)	1966	2.93	2.98	3.10	3.20	3.09	−0.033
	1975	2.59	2.68	2.86	2.88	2.79	
Forced vital capacity (litres)	1966	3.87	3.97	4.04	4.17	4.06	−0.020
	1975	3.61	3.69	3.97	4.04	3.88	
FEV_1/FVC (%)	1966	76.0	77.2	77.4	76.5	76.7	−0.62
	1975	71.7	70.6	71.9	71.1	71.1	
Residual volume (litres)	1966	1.66	1.53	1.76	1.79	1.70	+0.032
	1975	1.93	1.90	2.06	2.04	1.99	
Total lung capacity (litres)	1966	5.74	5.57	6.10	6.25	5.98	+0.011
	1975	5.71	5.76	6.31	6.31	6.08	
RV/TLC (%)	1966	28.9	27.4	28.8	28.6	28.3	−0.49
	1975	33.6	32.8	33.4	32.2	32.7	
Transfer factor (mmol/min/kPa)	1966	8.28	8.55	9.07	9.30	8.94	−0.041
	1975	7.69	8.24	8.74	8.94	8.57	

Lung function results standardized to the overall average age and height in each year

Forced vital capacity was higher and declined least among those in occupation groups III and IV. The 27 men who gave up smoking between 1966 and 1975 had lower values than either those who continued smoking or who had already given up. The 7 men with both persistent crackles and wheezes in 1966 showed a decline of over 90 ml per year, four times the expected decline. The variation of forced vital capacity with radiographic abnormality was rather less, although the lowest average values occurred for those with small opacities of category 1/1 or more, or with diffuse pleural thickening.

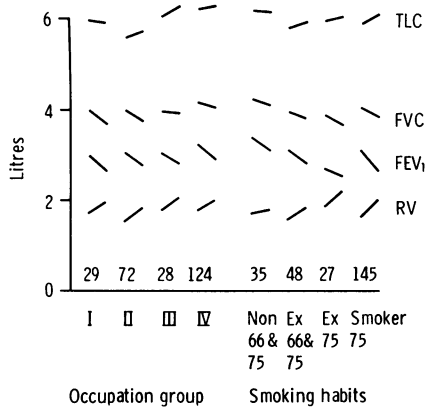

Figure 1. Lung volumes and flow rates: changes between 1966 and 1975 by occupation group in 1966, and smoking habits in 1966 and 1975 (each line joins the average value in 1966, standardized to age 42 and height 1.706 metres, to the average value in 1975, standardized to age 51 and height 1.709 metres; numbers of subjects are shown on the *x* axis)

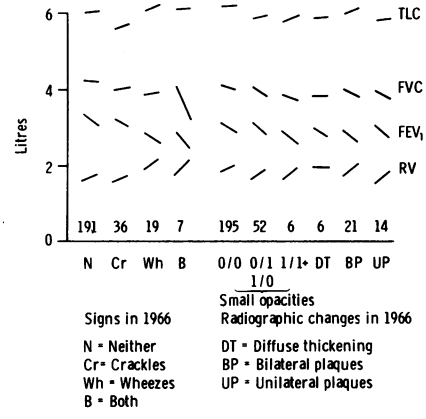

Figure 2. Lung volumes and flow rates: changes between 1966 and 1975 by physical signs in 1966 and radiographic appearances in 1966

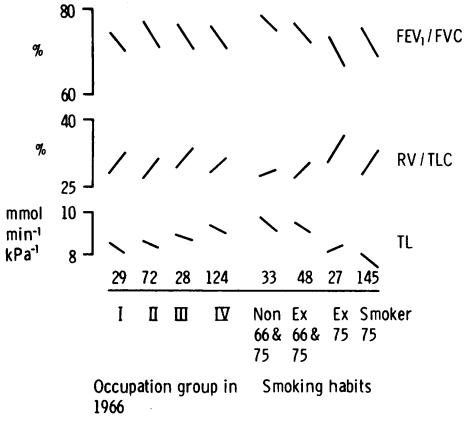

Figure 3. Volume ratios and gas transfer: changes between 1966 and 1975 by occupation group in 1966, and smoking habits in 1966 and 1975

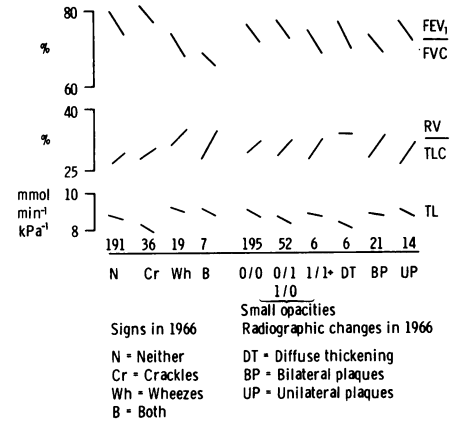

Figure 4. Volume ratios and gas transfer: changes between 1966 and 1975 by physical signs in 1966 and radiographic appearances in 1966

Forced expiratory volume showed patterns similar to those for forced vital capacity, except that the average values were lower for all 26 men who had chest wheezes in 1966, rather than just for those with both crackles and wheezes.

Residual volume was lower in occupation group II than in the other groups. It was markedly raised and showed the greatest rises in those who smoked in 1966 irrespective of whether they had subsequently given up smoking. It was also raised in those men with chest wheezes or with diffuse pleural thickening in 1966.

The FEV_1/FVC and RV/TLC ratios showed complementary results (Figures 3 and 4) with worse average values for those who smoked and for those with wheezes in 1966. The 6 men with diffuse pleural thickening showed no rise in RV/TLC ratio between 1966 and 1975, but there were too few men in this group to permit firm conclusions to be drawn.

The transfer factor of the lung showed greater differences than did any other index. There was a consistent trend with occupation group, being lowest in group I and highest in IV. Smokers had much lower values than did non- and ex-smokers. The 27 men who gave up smoking between 1966 and 1975 showed an average rise over this time as would be expected because of the immediate effect of smoking on transfer factor. Gas transfer was also reduced among those with persistent crackles and among those with diffuse pleural thickening.

Mortality amongst the study population

The numbers are small, and the time scale (9 years) too short to draw firm conclusions from the pattern of mortality occurring in the study population between the surveys. The numbers and causes of deaths occurring in the total group of 420 men in the original study are shown in Table 8. More than the expected number of deaths occurred in the three occupation groups

Table 8. Causes of deaths between 1966 and 1975 among 420 men in original study

Occupation group	Asbestosis	Mesothelioma	Lung cancer	Total deaths (all causes)	Expected deaths (all causes)
I	2	1	2	13	9.7
II	3	2	4	14	9.7
III	1	0	1	8	4.9
IV	0	0	1	14	19.2
Total	6	3	8	49	43.5

most exposed to asbestos. In occupation group I, three of the thirteen deaths were definitely associated with asbestos, while asbestos might also have played a part in the two who died of lung cancer. Seven of the 14 deaths in laggers (group II) were associated with asbestos, while exposure to the dust might have played a part in four more of the deaths. There were no deaths directly attributable to asbestos in the least exposed group IV.

Discussion

The findings of the original 1966 cross-sectional study of 420 men at Devonport resulted in major improvements in working conditions and the introduction of substitute materials for asbestos which, since 1968, has led to the virtual cessation of exposure to asbestos for all except a small number of men engaged in removing existing asbestos insulating materials from ships. Even these men can now be regarded as unexposed because of the stringent precautions enforced for their respiratory protection (Harries 1971).

Despite these improvements a very small risk may possibly exist from time to time through failure of the preventive measures or because of the uncertain effects on health of the new substitute insulating materials. There is also a need to detect changes in men working with these materials, so that they may be advised and removed from further risk if they show early stages of respiratory disease.

This study was designed to examine and compare among the survivors of the population chosen for the original study the clinical, radiographic and lung function indices in relation to exposure patterns and progression of disease.

Smoking affects the absolute values and rates of changes of lung function indices, but, just as in the initial survey, there were no significant differences in smoking habits between occupation groups. However, there was a significant relation between smoking habit and the presence of chest wheeze and between smoking habit and the development of both parenchymal and pleural radiographic abnormalities.

Analysis of the lung function patterns in relation to occupation group, smoking habit, presence in 1966 of persistent crackles and chest wheezes, and to radiographic appearance in 1966, showed that the transfer factor of the lung differentiated most sharply between the occupation groups and also related strongly to smoking habit and to the presence of persistent crackles.

Those with persistent crackles also had a reduced total lung capacity suggesting the development of a restrictive pattern of lung function, whereas those with wheezes had reduced forced vital capacity, forced expiratory volume and raised residual volume: all aspects of obstructive lung disease.

Total lung capacity, forced vital capacity and forced expiratory volume were all somewhat lower among those few men with definite parenchymal radiographic abnormalities, but the greater functional reduction occurred among the 6 men with diffuse pleural thickening in 1966. These had reduced transfer factor and raised residual volume as well as low values for total lung capacity and forced vital capacity.

References

Becklake M R, Fournier-Massey G, McDonald J C, Siemiatycki J & Rossiter C E (1970) *Bulletin de Physio-Pathologie Respiratoire* **6**, 637–659

Cotes J E (1965) Lung Function. 2nd edn. Blackwell, London & Edinburgh; p 604

Elmes P C (1966) *Postgraduate Medical Journal* **42**, 623–635

Harries P G (1970) The effects and control of diseases associated with exposure to asbestos in a Naval Dockyard. MD Thesis, University of London

Harries P G (1971) *Annals of Occupational Hygiene* **14**, 241–254

International Labour Office (1972) ILO U/C international classification of radiographs of pneumoconioses 1971. Occupational Safety and Health Series 22 (rev). ILO, Geneva; p 37

Lumley K P S L (1977) In: Inhaled Particles IV. Ed. W H Walton. Pergamon Press, Oxford; pp 781–788

Rossiter C E & Harries P G (1979) *British Journal of Industrial Medicine* **36**, 281–291

Selikoff I J, Churg J & Hammond E C (1965) *Annals of the New York Academy of Sciences* **132**, 139–155

Sheers G & Templeton A R (1968) *British Medical Journal* iii, 574–579